

At an Increased Risk: Tumor Lysis Syndrome

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Patients at highest risk for tumor lysis syndrome (TLS) often are diagnosed with bulky, rapidly proliferating hematologic tumors, such as acute leukemia and non-Hodgkin lymphoma (Kaplow & Hardin, 2007). Patients with solid tumors, such as mediastinal masses which are highly sensitive to chemotherapy, also may develop TLS, although it is more common in patients undergoing treatment for leukemia and lymphoma. TLS occurs from the effect of chemotherapy or radiation on rapidly dividing cells. Patients with elevated lactic dehydrogenase (LDH), dehydration, and renal insufficiency are at greatest risk for developing TLS (Brant, 2002). Advances in cancer treatment, such as those in bone marrow transplantation, require the use of high-dose chemotherapy, which may demonstrate an increase in the incidence of TLS.

TLS is a rapidly developing oncologic emergency characterized by electrolyte and metabolic disturbances that are fatal without timely identification and management. Patients present with hyperuricemia, hyperphosphatemia, hypocalcemia, and hyperkalemia. Electrolyte disturbances can cause acute renal and multisystem organ failure (see Figure 1).

Pathophysiology

TLS occurs from the rapid release of intracellular components during cell death. Cancer cells have an abnormally high amount of potassium, phosphorus, and nucleic acid (Kaplow & Hardin, 2007). When cancer cells are destroyed by chemotherapy or radiation, they spill their intracellular components into the bloodstream, causing an influx of potassium, phosphorus, and nucleic acid which the kidneys are not able to efficiently excrete, leading to hyperuricemia,

hyperphosphatemia, hypocalcemia, and hyperkalemia. Hyperuricemia occurs when the liver converts nucleic acids into uric acid; hypocalcemia develops as serum calcium binds to elevated amounts of phosphorus within the bloodstream (Kaplow & Hardin).

Clinical Findings

Laboratory findings will demonstrate electrolyte imbalances such as hyperuricemia (more than 6.0 mg/dl), hyperphosphatemia (more than 4.5 mg/dl), hypocalcemia (less than 8.5 mg/dl), and hyperkalemia (more than 5.5 mEq/l) (McCance & Heuther, 2006). Hyperkalemia generally is the first electrolyte imbalance, followed by hyperphosphatemia, and leads to hypocalcemia and hyperuricemia (Agnani, Gupta, Atray, & Vachharajani, 2006). Multisystem organ failure may occur because of these metabolic findings. TLS has the ability to affect the renal, gastrointestinal, cardiac, and neuromuscular systems.

Renal

Hyperuricemia occurs when uric acid crystals and calcium phosphate salts obstruct renal flow, causing renal failure (Kaplow & Hardin, 2007). Patients will have increased uric acid and creatinine levels, indicating renal failure. Physical symptoms consist of flank pain, gross hematuria, cloudy urine, oliguria, lethargy, nausea, and vomiting (Murphy-Ende & Chernecky, 2002). Weight gain and edema also may be present.

Gastrointestinal

Hyperkalemia causes nausea, vomiting, and diarrhea (Cope, 2004). Anorexia, abdominal cramping, and pain also may occur because of the elevated potassium (McCance & Heuther, 2006). Decreased levels of serum calcium may cause intestinal cramping and increased bowel activity (McCance & Heuther).

Cardiac

Serum potassium levels more than 5.3 mEq/l may cause irregular heart arrhythmias and hypotension (Murphy-Ende & Chernecky, 2002). Often the dysrhythmias are atrial in origin (Kaplow & Hardin, 2007). Hyperkalemia causes electrocardiogram changes such as tall T waves, flattened P waves, prolonged PR intervals, widened QRS complexes, and depressed ST segments (Murphy-Ende & Chernecky). The presence of a prolonged QT interval may indicate hypocalcemia.

Neuromuscular

Hyperkalemia causes neuromuscular irritability, muscle weakness, or paralysis, and hypocalcemia causes neuromuscular excitability (McCance & Heuther, 2006). A decreased level of serum calcium also may cause seizures and tetany (McCance & Heuther).

Medical Management

To prevent morbidity and mortality, early recognition and management of TLS are of primary concern. Patients at

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